Insect Vector Interactions with Persistently Transmitted Viruses*

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Abstract

The majority of described plant viruses are transmitted by insects of the Hemipteroid assemblage that includes aphids, whiteflies, leafhoppers, planthoppers, and thrips. In this review we highlight progress made in research on vector interactions of the more than 200 plant viruses that are transmitted by hemipteroid insects beginning a few hours or days after acquisition and for up to the life of the insect, i.e., in a persistent-circulative or persistent-propagative mode. These plant viruses move through the insect vector, from the gut lumen into the hemolymph or other tissues and finally into the salivary glands, from which these viruses are introduced back into the plant host during insect feeding. The movement and/or replication of the viruses in the insect vectors require specific interactions between virus and vector components. Recent investigations have resulted in a better understanding of the replication sites and tissue tropism of several plant viruses that propagate in insect vectors. Furthermore, virus and insect proteins involved in overcoming transmission barriers in the vector have been identified for some virus-vector combinations.

INTRODUCTION

The Majority of Recognized Plant Virus Species are Vectored by Hemipteran Insects

Hemipteran: insect belonging to the order Hemiptera that includes aphids, whiteflies, leafhoppers, planthoppers, and true bugs, but not thrips

Stylets: needle-shaped mouthparts of insects, mites, and nematodes

Insects transmit the majority of described plant viruses. Of the 697 virus species recognized by the International Committee on Taxonomy of Viruses (ICTV) (**http://phene.cpmc.columbia.edu/Ictv/index.htm**, last modified on 08/12/07), insects and other vectors transmit 76% (**Table 1**). In addition to these 697, we estimate that there are over 400 plant viruses that have been reported but are not yet officially recognized by the ICTV most of which have not been included in the tables. Hemipteran insects transmit the majority of the vectored viruses (55%) (**Table 1**).

Hemipteran insects have distinct features that allow for efficient virus transmission. The predominant feature of these insects is that they have piercing-sucking mouthparts that include a needle-like stylet bundle consisting of two mandibular and two maxillary stylets (40). The two maxillary stylets are interlocked and between them form two canals. The narrower salivary canal delivers saliva into the feeding puncture in plant tissues and the wider food canal takes up plant sap into the cibarium (the sucking pump), the esophagus, and the rest of the alimentary canal. Some plant-feeding hemipteran insects are specialized as phloem, xylem, or mesophyll feeders, whereas others can feed on a combination of these tissues (34). Similarly, many plant viruses transmitted by hemipteran insects are phloem-limited, whereas others are not tissue specific and exploit almost all plant tissues.

The plant-feeding hemipteran insects were formerly classified in the suborder Homoptera, which included aphids (Aphidoidea), whiteflies (Aleyrodoidea), jumping plant lice (Psylloidea), scale insects (Coccoidea) belonging to the Sternorrhyncha, and leafhoppers (Cicadelloidea), planthoppers (Fulgoroidea), froghoppers/spittlebugs (Cercopoidea), and cicadas (Cicadoidea) belonging to the Auchenorrhyncha (70). However, phylogeny based on molecular data and reinterpretation of morphological characteristics revealed that the Homoptera,

Table 1 Vectors and the plant viruses that they transmit

Vector taxa	Vector group	Virus groups				Total	%
		Icosahedral particles RNA genome	Rod-shaped particles RNA genome	DNA genome	Enveloped particles RNA genome		
Hemiptera	Aphids	26	153[a]	13	5	197	28
	Whiteflies	–	13	115[b]	–	128	18
	Leafhoppers	8	–	15	3	26	4
	Planthoppers	10	4[c]	–	4	18	3
	Other hemiptera	–	8	5	–	13	2
Thysanoptera	Thrips	2	–	–	14	16	2
Coleoptera	Beetles	50	1	–	–	51	7
Acari	Mites	10	9	–	–	10	1
Nematoda	Nematodes	45	3	–	–	48	7
Mycota	Fungi	8	16	–	–	24	3
	No identified vectors	84	60	19	3[d]	166	24
	Total	233	268	167	30	697	
	%	33	39	24			

[a]Includes 110 virus species of the genus *Potyvirus*, family *Potyviridae*; [b]Virus species of the genus *Begomovirus*, family *Geminiviridae*; [c]These are all tenuiviruses that have multiple shapes; [d]These viruses probably have insect vectors.

particularly the Auchenorrhyncha, are not monophyletic (32, 70, 159, 182) even though some morphological characteristics suggest monophyly (196). In a revised phylogeny the Auchenorrhyncha has been split into two suborders: (*i*) the planthoppers (Archaeorrhyncha or Fulgoromorpha) and (*ii*) leafhoppers, treehoppers, spittlebugs, and cicadas (Clypeorrhyncha or Cicadomorpha) (32, 159). These two suborders together with the suborder Prosorrhyncha, which includes the Heteroptera, form the Euhemiptera to which the suborder Sternorrhyncha is a monophyletic sister group (40, 71).

The most economically important insect vectors are restricted to a few hemipteran families. These are the Aphididae (aphids) and Aleyrodidae (whiteflies) of the suborder Sternorrhyncha, Cicadellidae (leafhoppers) of the suborder Cicadomorpha, and Delphacidae (delphacid planthoppers) of the suborder Fulgoromorpha (126). Aphids and whiteflies transmit 325 plant virus species (**Table 1**). These include 110 virus species of the genus *Potyvirus* that are solely transmitted by aphids, and 115 virus species of the genus *Begomovirus* that are solely transmitted by whiteflies (**Table 1**). Leafhoppers transmit 26 plant viruses and delphacid planthoppers 18 plant viruses (**Table 1**). Finally, 13 plant viruses are transmitted by other hemipteran insects (**Table 1**) including scale insects, jumping plant lice, and treehoppers.

Thrips (order Thysanoptera) are close relatives of hemipteran insects; they both belong to the Hemipteroid assemblage along with the lice (ectoparastites of birds and mammals in the order Phthiraptera) and plant lice (order Psocoptera) (40). The thrips mouthparts are composed of two maxillary stylets and one mandibular stylet that form a feeding apparatus (40).

Mites are closely related to the spiders and ticks. Eriophyid mites (Eriophyidae) and flat mites (Tenuipalpidae) are transmitters of plant viruses (96, 135, 158, 162). They have needle-like stylet structures that can puncture cells at the surface inducing leaf deformations (37, 140).

The plant-associated dagger (*Xiphinema* spp.), needle (*Longidorus* spp.), and "stubby-root" nematodes (*Trichodorus* spp.) transmit several plant viruses (30). These nematodes have stylets, often referred to as spears or daggers, which can be long and sometimes reach the plant phloem.

Hemipteran Insects and Thrips Transmit Virus Species by Different Mechanisms

There are currently four described mechanisms of insect transmission of plant viruses. Originally, Watson & Roberts proposed a classification of plant viruses with regards to transmission into two groups, nonpersistent and persistent (183). This classification was restricted to viruses transmitted by insects of the Hemipteroid assemblage. In nonpersistent transmission, insects can inoculate the virus into plants for only a few minutes after acquisition and the insect loses the virus within a few minutes and upon molting. In persistent transmission, insects can inoculate the acquired virus for much longer periods (days/week), transmitting the virus after molting and often for their entire lifespan (larvae or/nymphs into adults).

Later, it was recognized that an intermediate category of semipersistent viruses exist; these can be transmitted by the vector from a few hours to a few days post acquisition but are lost after molting (93, 166) (**Tables 2**, **3**). Based on observations that nonpersistent viruses are retained by the vector mainly in the stylet (food canal) (11, 93), whereas semipersistent viruses are retained mainly in the foregut (127), Nault (126) used the terms nonpersistent stylet-borne and semipersistent foregut-borne viruses for these two categories. However, recently it was revealed that the semipersistently transmitted virus *Cauliflower mosaic virus* (CaMV) is retained in the stylet (178). Thus, biological transmission characteristics are not always strictly correlated with location of virus in the insect vector.

Non- and semipersistent viruses do not require a latent period, which is the time between the acquisition access period (AAP) and inoculation access period (IAP) (126). Furthermore,

Hemipteroids: insects that include hemipterans and their close relatives, i.e., thrips, lice and plant lice

Nonpersistent viruses: plant viruses for which inoculativity by the vector is retained for only a few seconds/minutes after acquisition from plants and is also lost after molting

Semipersistent viruses: plant viruses for which inoculativity by the vector is retained for a few hours to a few days after acquisition from plants but vectors lose upon molting

Foregut: anterior part of the alimentary canal

Latent period: the time between acquiring the virus by the vector and the ability to transmit it

Table 2 Transmission characteristics and timing of plant viruses transmitted by hemipteran insects

Biological characteristic	Nonpersistent stylet-borne	Semipersistent foregut-borne[b]	Persistent circulative	Persistent propagative
AAP and IAP[a]	Seconds, minutes[c]	Minutes, hours[d]	Hours, days[d]	Hours, days[d]
Latent period	None	None	Hours, days	Days, weeks
Retention time in vector	Minutes, lost after molting	Hours, lost after molting	Days, weeks	Lifespan of insect
Presence in vector's hemolymph	No	No	Yes	Yes
Multiplication in vector	No	No	No[e]	Yes
Transovarial transmission	No	No	No	Often

[a]AAP, Acquisition access period; IAP, Inoculation access period; [b]A recent publication revealed that the semi-persistent virus *Cauliflower mosaic virus* (CaMV) is retained in the stylet (178); [c]The time period during which virus can be acquired from and inoculated into plant epidermal cells; [d]AAP and IAP times depend on the location of the virus in the plant, i.e., acquisition of the virus from the plant phloem takes longer than acquisition from the epidermis or mesophyll cells; [e]Except for TYLCV for which there is evidence that it replicates in its whitefly vector.

Hemolymph: insect equivalent of blood

Hemocoel: body cavity of insects that contains the hemolymph and all internal organs

Persistent viruses: plant viruses for which inoculativity by the vector is retained for long periods (days to weeks), often throughout the vector's lifespan, and also is retained after molting

these viruses cannot be recovered from the insect hemolymph and cannot be transmitted to plants upon injection into the hemocoel of the insect vector. The semipersistent viruses generally have longer AAPs and IAPs than the nonpersistent viruses (**Table 2**), presumably because most of the former viruses are phloem-limited, whereas the latter are non-tissue specific (**Figure 1**).

Many of the non- and semipersistently transmitted viruses, e.g., potyviruses and caulimoviruses, need one or more helper components for transmission. These helper components are proteins produced by the virus during plant infection and are required for attachment of virus particles to the inner cuticular lining of the vector's maxillary stylets, particularly near the tip where the food and salivary canals merge, via a "bridge structure" (reviewed in 130). This attachment allows for temporary retention of virus particles inside the vector's food canal after which they are released into plants during feeding, probably through protease-mediated degradation of the attachment proteins. One of the two helper components of CaMV, the P2 protein, was shown to interact with a nonglycosylated protein receptor deeply embedded in the chitin matrix at the extreme tip of the stylet of the aphid vector (178). Other nonpersistent viruses, e.g., cucumoviruses, bind directly to the cuticular lining of the insect mouthparts without the need for helper component(s) (130).

The persistent viruses were later divided into two categories: the persistent circulative (mostly nonpropagative) viruses and the

Table 3 Modes of transmission of plant viruses by insect groups of the Hemipteroid assemblage

Vector taxa	Vector species	Modes of transmission				Totals	%
		NPV[a]	SPV[b]	PCV[c]	PPV[d]		
Hemiptera	Aphids	161[e]	19	12	5	197	49.4
	Whiteflies	5	9	115[f]	–	129	32.3
	Leafhoppers	–	4	13	10	27	6.7
	Planthoppers	–	–	–	18	18	4.5
	Other hemiptera	2	9	1	–	12	3.0
Thysanoptera	Thrips	2	–	–	14	16	4.0
	Totals	170	41	141	47	399	
	%	42.6	10.3	35.3	11.8		

[a]NPV, nonpersistent stylet borne viruses; [b]SPV, semipersistent foregut-borne viruses; [c]PCV, persistent circulative (mostly nonpropagative) viruses; [d]PPV, persistent propagative viruses; [e]Includes 110 virus species of the genus *Potyvirus*, family *Potyviridae*; [f]virus species of the genus *Begomovirus*, family *Geminiviridae*.

persistent propagative viruses (**Tables 2**, **3**) (126). Most of the circulative viruses apparently do not replicate in their insect vectors, whereas the propagative viruses do (**Figure 1**). Furthermore, the propagative viruses are often transmitted to the vector's progeny through infection of the embryos or germ cells in the female insects (64, 127, 167). For many persistently transmitted plant viruses, particularly in those infecting nonvegetatively propagated crops, insect transmission is obligatory for the plant virus, i.e., the insect vector is essentially the only means of virus spread in nature (126).

Persistent plant viruses move through the insect vector, from the gut lumen into the hemolymph or other tissues and finally into the salivary glands from which these viruses are introduced back into the plant during insect feeding. Persistent viruses, whether propagative of nonpropagative, can be transmitted to plants after injection into the insect hemocoel (167). In many cases, injected viruses are transmitted at higher rates than orally acquired viruses, because movement of the virus across the insect gut is often a significant barrier to transmission (10, 14, 120). Furthermore, these viruses may multiply or accumulate in the insect vector without being transmitted. Indeed, only 10%–34% of *Dalbulus maidis* leafhoppers exposed to plants infected with *Maize rayado fino virus* (MRFV; *Marafivirus*) transmitted virus, although ca 80% contained virus as determined serologically (58), and only 9% of the planthopper *Peregrinus maidis* transmitted *Maize stripe virus* (MStV; *Tenuivirus*), although the virus was detected by ELISA in 23% of the planthoppers that had access to infected plants (129).

Four types of barriers to persistent transmission of plant and animal viruses in their vectors have been proposed or identified (reviewed in 5, 76): (*i*) midgut infection barrier, (*ii*) dissemination (including midgut escape and salivary gland infection) barriers, (*iii*) salivary gland escape barrier, and (*iv*) transovarial transmission barriers. Passage of persistent viruses through different organs in their insect vectors requires specific interactions between virus and vector components (68, 80, 138, 192). These components have been identified for a number of plant viruses. In this review we highlight progress made in research on vector interactions of viruses that are transmitted in a persistent manner by hemipteran insects and thrips (**Tables 3**, **4**, and **5**; **Figure 1**).

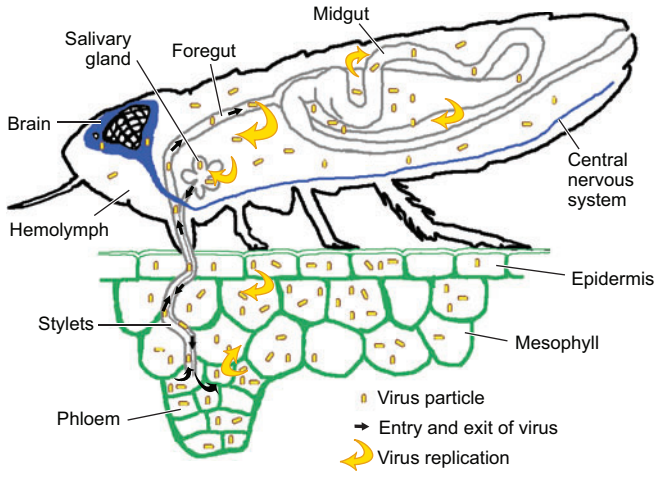

Figure 1

Schematic representation of persistent virus transmission by a leafhopper. Viruses that are transmitted in a circulative persistent manner do not replicate in the insect and usually enter the salivary glands from the hemolymph. In plants, replication of circulative viruses is frequently restricted to the phloem tissues. In contrast, most propagative viruses replicate in several plant tissues and in different organs of the insect vectors (*yellow arrows*) and may enter the salivary glands either from the hemolymph or from other connecting tissues, e.g., the nervous system or trachea. In contrast, the nonpersistent and semipersistent viruses attach to the inner cuticular linings of the insect vector stylet and foregut either directly or indirectly via helper components, and are introduced into plants during insect salivation and regurgitation (reviewed in 130).

VIRUSES TRANSMITTED IN A PERSISTENT-CIRCULATIVE MANNER

Virus species of the *Luteoviridae*, *Geminiviridae* and *Nanoviridae* families are transmitted in a persistent circulative manner (**Table 4**; **Figure 1**). All these viruses have icosohedrical particles in which the nucleic acids are contained within in a proteinaceous capsid without a lipid envelope. Luteoviruses and nanoviruses are transmitted solely by aphids, whereas the geminiviruses can be transmitted by whiteflies or leafhoppers, and the single *Topucovirus*

Propagative viruses: viruses that invade and replicate in various tissues of their vectors

Circulative viruses: viruses that move from the gut into the hemolymph and other tissues of their vectors

Transovarial transmission: transmission of viruses from female parent to offspring through the ovaries

Table 4 Viruses transmitted in a persistent circulative manner by hemipteran insect groups

Virus family	Virus genus	Number[a]	Hemiptera			
			Aphids	Leafhoppers	Whiteflies	Treehopper[a]
Luteoviridae	*Enamovirus*	1	1	–	–	–
	Luteovirus	2	2	–	–	–
	Polerovirus	5	5	–	–	–
Geminiviridae	*Mastrevirus*	10	–	10	–	–
	Curtovirus	3	–	3	–	–
	Begomovirus	115	–	–	115	–
	Topocuvirus	1	–	–	–	1
Nanoviridae	*Nanovirus*	3	3	–	–	–
	Babuvirus	1	1	–	–	–
TOTALS		141	12	13	115	1

[a]Classified as "other" in **Tables 1** and **3**; –, No vectors identified.

species, *Tomato pseudocurly top virus* (**http://phene.cpmc.columbia.edu/index.htm**), is transmitted by the treehopper *Micrutalis malleifera* (Hemiptera: Membracidae) (**Table 4**). We prefer to use the term circulative rather than the previously and widely used term circulative nonpropagative viruses, because some of these viruses may be shown later, by more sensitive techniques, to replicate in their vectors, as is the case with *Tomato yellow leaf-curl virus* (TYLCV; genus *Begomovirus*; family *Geminiviridae*) (described below).

Luteoviruses

The family *Luteoviridae* consists of the genera *Enamovirus*, *Luteovirus*, and *Polerovirus* (52). These three genera have slightly different genome organizations. In addition, the single member of the genus *Enamovirus*, *Pea enation mosaic virus* 1 (PEMV1), cannot systemically move throughout the plant without the presence of a second virus, *Pea enation mosaic virus* 2 (PEMV2), that belongs to the genus *Umbravirus*. The PEMV viruses infect plant cells

Table 5 Viruses transmitted in a persistent propagative manner by various hemipteroid insect groups

Virus family	Virus genus	Number	Hemiptera			Thrips
			Aphids	Leafhoppers	Planthoppers	
Bunyaviridae	*Tospovirus*[a]	14	–	–	–	14
Rhabdoviridae	*Cytorhabdovirus*[a]	8[b]	3	1	2	–
	Nucleorhabdovirus[a]	11[b,c]	2	3	2	–
	Unassigned[a]	1[c]	–	–	–	–
Reoviridae	*Fijivirus*	8	–	–	8	–
	Oryzavirus	2	–	–	2	–
	Phytoreovirus	3	–	3	–	–
Tymoviridae	*Marafivirus*	3	–	3	–	–
Unassigned	*Tenuivirus*[a]	4	–	–	4	–
TOTALS		41	5	10	18	14

[a]Enveloped viruses; [b]the insect vectors of three nucleorhabdovirus and two cytorhabdovirus have not yet been identified; [c]one nucleorhabdovirus, Coffee ringspot virus, and the unassigned Orchid fleck virus are transmitted by the mite *Brevipalpus phoenicis*; –, no vectors identified.

in various tissues, including epidermal cells, and can be introduced into plants by leaf-rub inoculation or aphid transmission, whereas members of the genera *Luteovirus* and *Polerovirus* are phloem-limited and dependent on aphids for transmission. Virus particles are nonenveloped icosahedral particles of ~30-nm diameter. The capsid consists of two coat proteins: the major coat protein of 22 kDa and a minor readthrough (RT) protein of 76 kDa. The RT protein contains the 22-kDa major coat protein amino acid sequence at its amino end and a 54-kDa readthrough domain (RTD) at the C terminus. The RTD protrudes from the exterior of virions.

Luteoviruses are likely to enter the epithelial cells of the vector's gut by endocytosis and exit these cells to enter the hemocoel by exocytosis (59, 68). The particles can move rapidly through the intestinal cell layer, i.e., they are observed inside these cells at 4 h after acquisition and can enter the hemocoel at 8 h after acquisition (60). Virus particles accumulate at high numbers in epithelial cells when aphids feed from a virus source. However, when aphids are removed from the virus source, virus content in these cells starts to decrease rapidly after the second day (60). Transport of virus through the salivary gland cells also involves endocytosis and exocytosis. The various barriers that luteoviruses encounter in aphid vectors have been reviewed by Gray & Gildow (68).

Both the CP (coat protein) and RTD have been implicated in aphid transmission (25, 27, 31, 98). The RTD also determines virus-gut tropism in the aphid, i.e., the capability of the viruses to invade only midguts or both midguts and hindguts is driven by the RTD (26). The CP and RT proteins of *Beet western yellows virus* (BWYV) are glycosylated and aphid transmission efficiency is inhibited by α-D-galactose-specific lectin or α-D-galactosidase treatment of BWYV particles (152). This finding led to the hypothesis that the glucidic core plays a direct role in the virus entry into gut epithelial cells. However, glycosylation may also have an indirect role in preventing virus degradation through, for example, interaction with the chaperonin symbionin (152). Symbionin is a homologue of the chaperonin GroEL and is abundantly produced by the bacterial endosymbiont, *Buchnera aphidicola*, of aphids (88). Symbionin directly binds luteovirus particles (153, 180) and protects them from degradation in the hemolymph, but does not mediate insect vector specificity (179). Residues in the equatorial domain of symbionin bind to the RTD of the minor capsid protein of luteovirus particles (53, 81, 82, 153, 179). Two symbionin molecules occur in aphids, a protein of 65 kDa that corresponds to the predicted size of the full-length symbionin monomer that is only present in the soluble protein fraction of aphid extracts and an apparently truncated protein of ~52 kDa that is present in soluble and membrane protein fractions (153). Indeed, a symbionin of ~56 kDa was isolated from gut brush border membrane vesicles of the mustard aphid, *Lipaphis erysimi*. When glycosylated, this symbionin binds mannose-binding garlic leaf lectin (19). Based on bioinformatic analyses, it was predicted that the mannose glycosylation sites that interact with the lectin are located in the symbionin equatorial domain (19) shown previously to be involved in luteovirus binding (81, 82). Thus, glycosylation of both virus coat proteins and symbionin may affect virus-symbionin interactions. With these new data, we are getting closer to elucidating the specific role(s) of symbionin in luteovirus transmission.

***Buchnera aphidicola*:** bacterial endosymbiont of aphids

Several luteovirus-binding aphid proteins, including new potential receptors, have been identified. The RTD of BWYV binds a *Drosophila melanogaster* Rack-1 homologue in the aphid *Myzus persicae* that is believed to play a key role in the endocytosis/transcytosis process of luteoviruses (153). Furthermore, whole BWYV particles bind the membrane-bound version of GAPDH3 in *M. persicae*. This GAPDH3 is proposed to be the receptor for BWYV in aphid midgut and accessory salivary gland (ASG) cells, because it regulates actin-dependent endocytosis and exocytosis in other organisms (153). The *Barley yellow dwarf virus* MAV (BYDV-MAV) isolate binds specifically to two proteins, SaM35 and SaM50, in the head

of its aphid vector *Sitobion avenae*, but does not bind proteins in the nonvector aphid *Rhopalosiphum maidis* (100). A recent study revealed that transmission of the polerovirus *Cereal yellow dwarf virus*—RPV (CYDV-RPV) by the aphid *Schizaphis graminum* is a heritable trait involving multiple genes regulating gut and salivary gland processes that are not genetically linked (195). Protein binding studies with parental and F_2 progeny of vector and nonvector genotypes of *S. graminum* showed that four proteins specifically bind CYDV-RPV particles in vector aphids. Two of the proteins were identified as luciferase and a cyclophilin, which are involved in macromolecular transport in cells in other organisms (195). Together, these results suggest that luteoviruses interact with different vector proteins for navigation through the gut and salivary gland transmission barriers.

Geminiviruses

The family Geminiviridae comprises four genera, *Mastrevirus*, *Curtovirus*, *Begomovirus*, and *Topocuvirus*. Geminivirus particle morphology and replication has been reviewed (72). Particle morphologies are typically geminate, appearing as twinned icosahedrial particles, ~18-X 30-nm dimensions. Their genomes consist of one or two single-stranded circular DNA molecules of ~3.0 kb in length. Species in these genera differ in vector specificity and genome organization. Mastreviruses are transmitted by leafhoppers and have monopartite circular DNA genomes that contain small and large intergenic regions. Curtoviruses are also transmitted by leafhoppers and have a monopartite genome that differs in organization from the mastreviruses. Begomoviruses are transmitted by whiteflies and, with a few exceptions, have bipartite genomes that are most complex among the geminiviruses. The genus *Topocuvirus* has only one member, *Tomato pseudocurly top virus* (TPCTV), which is transmitted by the treehopper *M. malleifera* (Hemiptera: Membracidae) (28). The TPCTV genome has features of both whitefly- and leafhopper-transmitted geminiviruses (28).

Maize streak virus (MSV) is the type species of the genus *Mastrevirus*. MSV is the most economically important and widespread disease of maize in sub-Saharan Africa, neighboring islands, Egypt, and Yemen (22). MSV is transmitted by nine leafhopper species in the genus *Cicadulina* that are endemic in Africa (22). MSV transmission efficiency depends on the leafhopper species, and the ability of a leafhopper to transmit MSV is a genetically heritable trait (22). Persistence of MSV in its leafhopper vector is very efficient; *C. mbila* individuals remained infective for 35 days after a 3-h acquisition access period on MSV-infected plants (146). Furthermore, the concentration of the capsid protein and the genome of MSV increase gradually in *C. mbila* individuals during their sustained feeding on MSV-infected maize (99, 146). Spatial distribution of MSV within *C. mbila*, monitored with quantitative PCR (99), showed that MSV accumulates mainly in the intestinal tract. This is consistent with the occurrence of large inclusions containing MSV particles in the epithelial cells of the filter chamber and midgut of vector insects (109; E.-D. Ammar, D. Gargani & M. Peterschmitt, unpublished information). In spite of the long virus retention and accumulation in *C. mbila*, no evidence of MSV multiplication in its vector has been obtained using hybridization (23), ELISA (146), or quantitative PCR (99).

The genus *Curtovirus* has only three virus species of which *Beet curly top virus* (BCTV) is the type member. BCTV is transmitted by the beet leafhopper *Circulifer tenellus* (Baker). The virus and insect vector have broad plant host ranges that include 300 plant species in 44 families. It is predominant in the western United States and the eastern Mediterranean.

The genus *Begomovirus* contains the majority of described species within the family *Geminiviridae*. Begomovirus interactions with whitefly vectors are complex. According to reports published so far, these viruses may or may not replicate in their vectors, may or may not be transovarially transmitted to next generation insects, and may or may not affect fecundity and longevity of insect vectors. *Tomato yellows leaf*

curl virus (TYLCV) apparently replicates in its vector (157) and reduces whitefly fitness (148, 157), whereas *Tomato mottle virus* (ToMoV) does not appear to replicate in whiteflies and does not affect whitefly fitness (157). TYLCV DNA was shown to be transmitted transovarially to the embryos/eggs of whiteflies (62) and can be sexually transmitted between these insects (61). However, Bosco et al. (21) found that *Tomato yellow leaf curl Sardinia virus* (TYLCSV) DNA but not TYLCV DNA was transovarially transmitted. Furthermore, the inherited TYLCSV DNA is not infectious, and therefore the transovarial transmission has no epidemiological relevance (21).

Others have reported that the fecundity and longevity of the B biotype whitefly can increase by up to 18- and 7-fold, respectively, when feeding on plants infected with *Tobacco curly shoot virus* (TbCSV) and *Tomato yellow leaf curl China virus* (TYLCCNV) (90). This is in sharp contrast to the harmful effects of TYLCV on whiteflies described above. In the experiments with TbCSV and TYLCCNV, the whitefly population density was up to 13 times higher on infected than on healthy plants after 56 days (90). Native *B. tabaci* did not show an increased performance on infected vs healthy plants (90). This may explain, at least in part, the invasive nature of B biotype *B. tabaci*, which has been responsible for the pandemic increase in begomovirus outbreaks (90). The begomoviruses have striking degrees of genetic diversity between and within virus species (138). This genetic diversity and the presence of subviral DNAs (161) could contribute to the different findings related to transovarial transmission, effect on whitefly fitness and replication in insect vectors of various groups.

The coat proteins of geminiviruses determine insect vector specificity (17, 29, 79, 133, 160) and are much less variable in sequence than geminivirus replication protein sequences (138). The specificity of leafhopper transmission of BCTV from insects to plants resides within the coat protein, because when the coat protein of *African cassava mosaic virus* (ACMV), which is a whitefly-transmitted begomovirus, is replaced with the BCTV coat protein, *C. tenellus* will transmit BCTV and the chimeric virus but not ACMV to *Nicotiana benthamiana* (29). Mutation analyses of the coat protein of another curtovirus, *Beet mild curly top virus* (BMCTV), which is also transmitted by *C. tenellus*, demonstrated that N-terminal amino acids 25-28 are important for insect transmission (160). It was suggested that this region of the coat protein is involved in receptor-mediated endocytosis in the gut and salivary glands of leafhoppers (160). Begomoviruses also require a functional coat protein for whitefly transmission (17, 102, 103). The composition of the coat protein from amino acids 123 to 149 and residues 149 to 174 contributes to whitefly transmission efficiency (83). Noris et al. (133) found that mutations in the amino acids at positions 129 and 134 of the TYLCV coat protein generate nontransmissible strains and that some naturally occurring nontransmissible TYLCV strains carry mutations at these sites.

Similarly to luteoviruses, begomovirus transmission by whiteflies depends on a GroEL homologue that carries structural similarities to the *Buchnera* symbionin and is produced by coccoid whitefly symbionts (115). TYLCV binds directly to this homologue of GroEL with a higher affinity than anti-TYLCV antibodies (2). Interference with the GroEL-geminivirus interaction in the whitefly hemolymph results in degradation of TYLCV particles (115). This finding led to novel applications immediately relevant to agriculture, such as the generation of TYLCV-resistant tomato plants that produce GroEL in the phloem (3). Note that the same host factors (i.e., two GroEL homologues) are involved in the transmission of viruses from two very different families and that both are transmitted in a persistent circulative manner. This suggests that GroEL has a basic and conserved role in transport of macromolecules in aphids and whiteflies.

Nanoviruses

The family *Nanoviridae* contains two genera: *Babuvirus* and *Nanovirus*. Morphology and

replication of nanovirus family members have been reviewed (69). Virus species in this family have small ~19-nm diameter icosahedral virus particles and carry multipartite (6 to 11 segments) ssDNA genomes. The ssDNA molecules are circular, ~1 kb in size, and encode mostly single proteins varying from 5 to 33 kDa in size. These viruses induce extreme stunting in plants. All nanoviruses are transmitted by aphids (**Table 4**).

The type member of the genus *Babuvirus* is *Banana bunchy top virus* (BBTV). Babuviruses consist of at least five monocistronic segments and one segment with two unidirectionally transcribed ORFs. The type member of the genus *Nanovirus* is *Faba bean necrotic yellows virus* (FBNYV). This virus is one of the most economically damaging disease agents of faba bean, causing up to 90% crop losses in Egypt and the nearby regions of Syria and Turkey. The disease spreads because aphid populations can survive the mild winters and provide a continuous inoculum source for FBNYV. Nanoviruses consist of at least eight monocistronic segments. FBNYV is efficiently transmitted by the aphid vector *Acyrthosiphon pisum*, but requires a helper component for transmission (55). This helper component is not only involved in virus acquisition but also appears to facilitate transport of viruses from the hemolymph into the salivary gland (55).

The subviral DNAs associated with geminiviruses are often nanovirus-like components that have adapted to whitefly transmission along with the begomoviruses (161). Furthermore, there is evidence that circoviruses, which infect vertebrates, evolved from nanoviruses through recombination and host-switch events (63). Thus, the nanovirus particles appear to adapt to and recombine easily with other virus species that have different hosts.

VIRUSES TRANSMITTED IN A PERSISTENT PROPAGATIVE MANNER

All enveloped plant viruses are transmitted in a persistent propagative manner (**Tables 3**, **5**; **Figure 1**). These virus species include bunyaviruses and rhabdoviruses (**Table 5**). Three groups of nonenveloped viruses, the reoviruses, tenuiviruses, and marafiviruses, are transmitted in a persistent propagative manner (**Table 5**). Most propagative viruses are also transmitted by a limited number of insect species/genera, e.g., leafhoppers, planthoppers, or aphids (**Table 5**). Thrips transmit all tospoviruses; leafhoppers transmit all marafiviruses and phytoreoviruses; and planthoppers transmit all fijiviruses, oryzaviruses, and all definitive tenuiviruses (**Table 5**). However, the two rhabdovirus genera are the exception to this rule, as various species of cytorhabdoviruses and nucleorhabdoviruses are transmitted either by aphids, leafhoppers, or planthoppers. Generally, within each vector species, certain populations/biotypes, different sexes, or different developmental stages (nymphs/adults) may differ in their ability to transmit the virus (126).

Tospoviruses

The genus *Tospovirus* belongs to the family *Bunyaviridae*. This family contains over 350 virus species divided into five genera: *Hanta-*, *Nairo-*, *Orthobunya-*, *Phlebo-*, and *Tospo-* viruses (reviewed in 131). The genus *Tospovirus* is unique within the *Bunyaviridae* because it contains plant-infecting viruses, whereas all other genera infect vertebrate animals. *Tomato spotted wilt virus* (TSWV) is the type member of the genus (54), which currently includes 14 recognized species. Within the genus *Tospovirus*, species are defined on the basis of the nucleocapsid (N) protein amino acid identity, vector specificity, and plant host range. Unique tospovirus species share less than 90% amino acid identity of the N protein. Many newly described viruses that are awaiting classification share characteristics with tospoviruses (52).

The structure of the TSWV virion is characteristic of members of the family *Bunyaviridae*, and like all viruses in this family, TSWV has a segmented, negative-sense and ambisense RNA genome (**Figure 2**). The virion measures

80-110 nm in diameter and incorporates an outer-membrane envelope derived from the host. Two glycoproteins (GPs) are embedded in the membrane and project from the surface. Three linear single-stranded RNAs, the S (2.9 kb), M (4.8 kb), and L (8.9 kb), are contained in the virion (41, 42, 97). The RNAs form pseudocircular structures that result from complementary base pairing at their ends and are found in association with a 29-kDa N protein (42). Virion particles also contain several copies of the 331.5-kDa RNA-dependent RNA polymerase (L) protein.

The thrips-tospovirus relationship is unique because adult thrips can only transmit TSWV if acquisition occurs in the larval stages (175). Adult thrips that feed on infected plants are unable to transmit virus even if they are allowed lengthy feeding periods on tospovirus-infected plants. Thrips are members of the insect order *Thysanoptera* (**Table 5**), and they are economically important as direct pests of crops and as vectors of plant viruses. Thrips that are efficient virus vectors are polyphagous, feeding on a wide array of plant species and plant organs. *Frankliniella occidentalis* (Pergande) is an efficient vector of tospoviruses, transmitting 5 of the 14 *Tospovirus* species and the TSWV-*F. occidentalis* interaction is the best-characterized tospovirus-vector interaction (reviewed in 192).

Most members of the *Bunyaviridae* are arthropod-borne and replicate in their vectors causing persistent, nonlethal, life-long infection. Within each genus, viruses are transmitted by a limited range of arthropod vectors. Unlike the infection of the vertebrate host, which is often characterized by an acute period of viremia, infection of the arthropod vector is persistent. Some *Orthobunyaviruses* are perpetuated by transovarial and sexual transmission between insect hosts (170, 184). The *Hantaviruses* are propagated within a single (or a few related) rodent species. *Hantaviruses* cause persistent asymptomatic infections within their natural rodent hosts, similar to infection in arthropod vectors belonging to other genera. Like *Hantaviruses*, *Nairoviruses* and *Phleboviruses* can be transmitted without an arthropod vector, but vector abundance and spread are the main causes of documented epizootics (104, 113, 193).

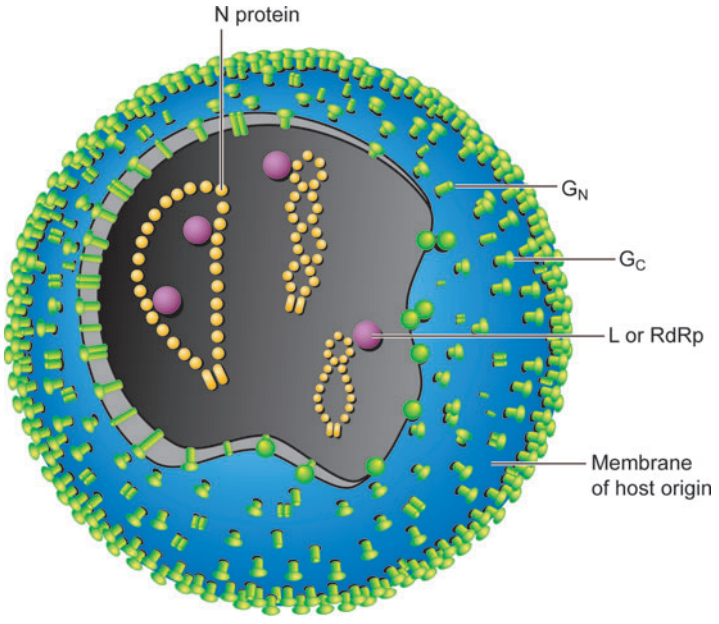

Figure 2

Diagram of TSWV virion. A double-layered membrane of host origin (*blue*) is shown with the viral-encoded proteins G_N and G_C (*green*) projecting from the surface in monomeric and dimeric configurations. The genomic RNA is presented as noncovalently closed circles in the form of a ribonucleoprotein (RNP) complex created by its association with many copies of N protein (*yellow*). A few copies of the virion-associated RNA-dependent RNA polymerase (RdRp or L) are shown (*purple*) in association with the RNPs. Graphic design by D.E. Ullman and E. Rendahl and reprinted from Reference 192 with permission from *Annual Review of Phytopathology*.

Tospoviruses encounter multiple tissue systems and membrane barriers along their path from the alimentary canal to the salivary glands in their thrips vectors (**Figure 1**). Upon ingestion of viral particles, virions travel through the lumen of the foregut into the midgut, the primary site of TSWV-binding and entry into insect cells (16, 120, 175). A brush border of microvilli extends into the midgut lumen and forms the first membrane barrier encountered by the virus. Virus particles move across the microvilli into the columnar epithelial cells of the midgut. Following replication in the epithelial cells, virions exit and traverse the basement membrane, the next barrier encountered by the virus. The midgut epithelium is encircled

by alternating series of longitudinal and circular muscle cells (116, 176). TSWV has been observed in these muscle cells, and entry and exit from these cells presumably constitute the third and fourth membrane barriers that virions must cross on their path to the salivary glands (16, 119, 120). The primary salivary glands are thought to play a critical role in virus acquisition and transmission. Tospoviruses entering the salivary gland must traverse the basal membrane of this tissue. The lumen of each primary salivary gland lobe is lined with microvilli, and this represents the last membrane the virus must cross for transmission to occur. Once inside the salivary gland lumen, virions can move with saliva into a canal that leads to an efferent salivary canal, a common salivary reservoir, and then a duct that ultimately allows virus-laden saliva to exit the combined salivary-food canal in the maxillary stylets.

Tospoviruses replicate within different tissue systems of the thrips. For example, the accumulation of nonstructural viral proteins (NSs) and viral inclusions composed of NSs in midgut epithelial cells, muscles surrounding the alimentary canal, and the primary salivary glands of thrips infected with TSWV provides direct evidence that viral replication occurs in these tissues (176, 177). In addition, Ullman et al. (177) found that TSWV glycoproteins were immunolocalized to thrips membranes thought to be part of the Golgi complex; in plants, the Golgi complex is the site of virion formation (95).

Studies of TSWV-thrips interactions and of other bunyaviruses provide evidence that the two surface-exposed glycoproteins play an essential role in the infection of insect vectors and animal cells. The two viral membrane glycoproteins, G_N and G_C, are encoded by the virion M RNA. The G_N and G_C are translated as a polyprotein from a single ORF (97). The resulting G_N/G_C polyprotein is cleaved to produce the individual glycoproteins that are required for virus transmission by thrips (1, 156, 189, 190). The two glycoproteins decorate the surface of the virion, and therefore are probably the first viral components that interact with molecules in the thrips midgut. A direct interaction between the G_N glycoprotein and thrips midguts has been demonstrated in vivo (190). Furthermore, exogenous G_N blocks virus acquisition and subsequent transmission by thrips vectors (189, 190). These findings provide evidence that G_N serves as a viral ligand and that mediates attachment of TSWV to receptors displayed on the epithelial cells of the thrips midgut. Additional evidence supporting a role for the glycoproteins in virus entry comes from studies of virus strains with reassorted genome segments. Isolates of TSWV that are serially mechanically passaged to plants accumulate mutations and deletions in the glycoprotein ORF, rendering these viruses nontransmissable by thrips (118, 121, 156). Work by Sin et al. (156) showed that the TSWV glycoproteins are necessary for virus infection of *F. occidentalis* and that a single point mutation in the glycoprotein ORF abolished transmission of the virus by insects. However, this alteration in the glycoprotein ORF did not compromise the ability of the virus to infect plants. Although there is evidence to support the role of G_N in virus attachment, recent evidence indicates that the TSWV G_C protein may mediate fusion of virion and cell membranes during entry into the insect vector cells (39, 191).

The identification of the virus receptors in thrips continues to elude scientists studying tospovirus-thrips interactions. Gel overlay assays of homogenized thrips and a thrips cDNA expression library have provided some candidate receptor proteins (18, 94, 112). The work of Bandla et al. (18) supports the hypothesis that G_N and/or G_C are involved in virus entry and interact with a receptor molecule in thrips. A 50-kDa protein in thrips (*F. occidentalis*) was identified as a candidate TSWV receptor by gel overlay analysis (18). A consistent difference in band intensity was observed between larval and adult thrips, a result that is compatible with known TSWV-thrips biology (i.e., efficiency of virus acquisition by larvae is reduced as the vector ages). Kikkert et al. (94) identified a 94-kDa protein in thrips that binds virus in the gel overlay assay, but this protein was not

present in the midgut of larval thrips and may be involved in virus specificity in other insect tissues. More research is needed to determine the specific involvement of the 50- and 94-kDa proteins in virus infection. The small size of thrips and lack of a cell culture system for thrips has limited the number of approaches that can be used to identify receptors. However, with the rapid improvement of proteomic technologies we expect that these barriers will be overcome in the coming years.

Rhabdoviruses

The *Rhabdoviridae*, a large virus family with members that infect vertebrates, invertebrates, and plants, includes pathogens of humans, livestock, and crops (56). The lyssaviruses that include *Rabies virus* infect primarily mammals and the novirhabdoviruses infect fish. Members of the remaining four genera, *Vesiculovirus*, *Ephemerovirus*, *Nucleorhabdovirus*, and *Cytorhabdovirus*, infect both a mammalian or plant host and an insect vector, and some vesiculoviruses also infect fish. The plant-infecting rhabdoviruses fall into two genera, *Nucleorhabdovirus* and *Cytorhabdovirus.* Unlike geminiviruses, cytorhabdoviruses and nucleorhabdoviruses cannot be distinguished based on insect vector group (**Table 5**), but are classified based on the intracellular localization of virus maturation and, more recently, on genome sequence (24). Cytorhabdoviruses mature in the cytoplasm of host plants on viroplasms in the endoplasmic reticulum, and nucleorhabdoviruses mature in the nucleus and bud through the nuclear membrane into the perinuclear space of host plant cells (14, 89).

Rhabdoviruses have distinct bullet-shaped particles that are easily recognized by electron microscopy, and this has led to the identification of more than 100 plant-infecting rhabdoviruses (89). However, a much smaller number of these rhabdoviruses has been characterized in any detail (89, 141). To date, complete sequence information is available for seven plant-infecting rhabdoviruses. Because there have been several recent reviews of plant infecting rhabdoviruses (80, 89, 141), we focus on recent results characterizing these viruses.

Plant rhabdoviruses, like other members of the *Rhabdoviridae*, have bacilliform virions of 45 to 100 nm in width and 130 to 350 nm in length. The genome of rhabdoviruses consists of a single, negative-sense genomic RNA encapsidated into nucleocapsid (N) protein subunits and surrounded by a lipid bilayer derived from plant or insect host cell membranes. The single viral glyco-(G) protein is embedded in the lipid membrane. The G protein molecules are exposed on the virion surface, making them easily visible in electron micrographs of virus particles (see (143). The matrix (M) protein interacts with both the nucleocapsid and lipid bilayer components of the virion. The negative polarity of the genome means that the mature virion must carry two other proteins, the phospho (P)-protein and large (L) protein, that are required for synthesis of viral mRNAs (89).

Rhabdoviral negative-sense RNA genomes are 12–14.5 kb in length, and encode 6 to 9 proteins. As for all rhabdoviruses, plant rhabdoviral genomes encode conserved leaders and trailers at the 5′ and 3′ ends of the genome, as well as conserved intergenic regions that are thought to be important for transcription initiation and termination (141, 169, 173). All known rhabdoviral genomes encode homologs for each of the five structural genes discussed above, exemplified by *Vesicular stomatitis virus* (VSV) (**Figure 3**). Each of the plant-infecting rhabdoviruses encodes at least one additional ORF, most commonly between the P and M genes (44, 85, 144, 145, 151, 169, 173, 188).

Functions are just beginning to be verified for the plant rhabdoviral genes homologous to VSV genes, and to be identified for the additional genes. The *Sonchus yellow net virus* (SYNV) *sc4* gene, *Lettuce necrotic yellows virus* (LNYV) gene *4b*, and the *Rice yellow stunt virus* (RYSV) gene *3* and Taro vein chlorosis virus (TaVCV) gene *3* encode predicted proteins with homology to the "30K" superfamily of proteins that allow virus movement in plants (44, 86, 114, 145). Further, expression of the RYSV gene 3-encoded protein in plants supported

Genus	Virus	Gene order	Reference
N	SYNV	N P *sc4* M G L	*
N	MMV	N P 3 M G L	Reed et al. 2006
N	TaVCV	N P 3 M G L	Revill et al. 2005
N	RYSV	N P 3 M G 6 L	Huang et al. 2003
N	MFSV	N P 3 4 M G L	Tsai et al. 2005
C	NCMV	N P 3 4 5 6 M G L	Tanno et al. 2000
C	LNYV	N P 4b M G L	Dietzgen et al. 2006
	VSV	N P M G L	

Figure 3

Genome organizations of various rhabdoviruses. Genus names: N, *Nucleorhabdovirus*; C, *Cytorhabdovirus*. Virus names: SYNV, *Sonchus yellows net virus*; MMV, *Maize mosaic virus*; TaVCV, Taro vein chlorosis virus; RYSV, *Rice yellow stunt virus*; MFSV, Maize fine streak virus; NCMV, *Northern cereal mosaic virus*; LNYV, *Lettuce necrotic yellows virus*; VSV, *Vesicular stomatitis virus*. Gene names: N, nucleocapsid protein; P, Phosphoprotein; M, matrix protein; G, Glycoprotein; L, Large protein encoding the viral RNA-dependent RNA polymerase. The positions and names of additional open reading frames (*sc4*, 3, 4, 4b, 5, and 6) encoded by specific virus are also indicated. References describing the viral genome sequences are indicated in the right column. *Many researchers contributed to the sequencing of this virus.

intercellular spread of a movement-deficient potexvirus (86). Using a binary plasmid designed for *Agrobacterium tumefaciens*–mediated expression of native and reporter-fusion proteins in plant cells, Goodin and coworkers (67) showed that the protein Sc4 is targeted primarily to the periphery of the cell, consistent with movement protein function. Because VSV is transmitted by and replicates in midges and flies, the five core genes of the rhabdoviral genome could be sufficient for replication of plant-infecting rhabdoviruses in their vectors. Nonetheless, the protein encoded by gene 6 of RYSV is expressed in insects (85).

The intracellular distribution of rhabdoviral proteins may provide clues about their function in plants and insects. Because nucleorhabdoviruses are assembled on nuclear membranes in plant and insect cells (12, 14, 89), transport of viral proteins synthesized in the cytoplasm into the nucleus is critical for virus replication. Both the SYNV and Maize fine streak virus (MFSV) N proteins carry a nuclear localization signal (NLS) (66, 141, 173), and both of these proteins accumulate in nuclei when expressed in *N. benthamiana* (66, 67, 173). Neither the SYNV nor the MFSV P proteins have a NLS, and both are found throughout the cell when expressed singly; however, they are found in subnuclear and nucleolar locations, respectively, when co-expressed with the N protein. The SYNV N and P proteins also interact in yeast two-hybrid experiments (66), suggesting that direct interactions between the two proteins are responsible for the shift in protein location.

Plant-infecting rhabdoviruses are transmitted in a persistent propagative manner, and some are transovarially transmitted. The leafhopper and planthopper transmitted rhabdoviruses are not transmitted by standard mechanical techniques to plants except with vascular puncture inoculation (VPI) (105), although the aphid-vectored rhabdoviruses can be mechanically transmitted by rub inoculation with some difficulty. Transmission efficiencies can range from as low as 5% to as high as 100% (5, 127, 168). Acquisition thresholds range from less than 1 min for rhabdoviruses that infect both mesophyll and phloem up to 15 min for phloem limited viruses. Inoculation thresholds range from a few minutes to several days. As with other viruses that replicate in their vectors, rhabdoviruses undergo a latent period of from 3 to more than 60 days.

Lyssaviruses and vesiculoviruses spread primarily through the nervous system of their vertebrate hosts (137, 149), and MMV (*Maize mosaic virus*) has been shown to infect most tissues in the vector *P. maidis*, including nervous tissue (12). This led to the hypothesis that

plant-infecting rhabdoviruses may also spread primarily through nervous tissue (80). Recent immunofluorescence microscopy studies of their insect vectors indicated that MMV is neurotropic in *P. maidis* (10) (**Figure 4**). Infection was more extensive and occurred much earlier in the nervous system than in most other tissues. A significantly higher proportion of planthoppers had infected midguts compared to those to infected salivary glands or to those that transmitted MMV, suggesting the occurrence of midgut and salivary gland barriers

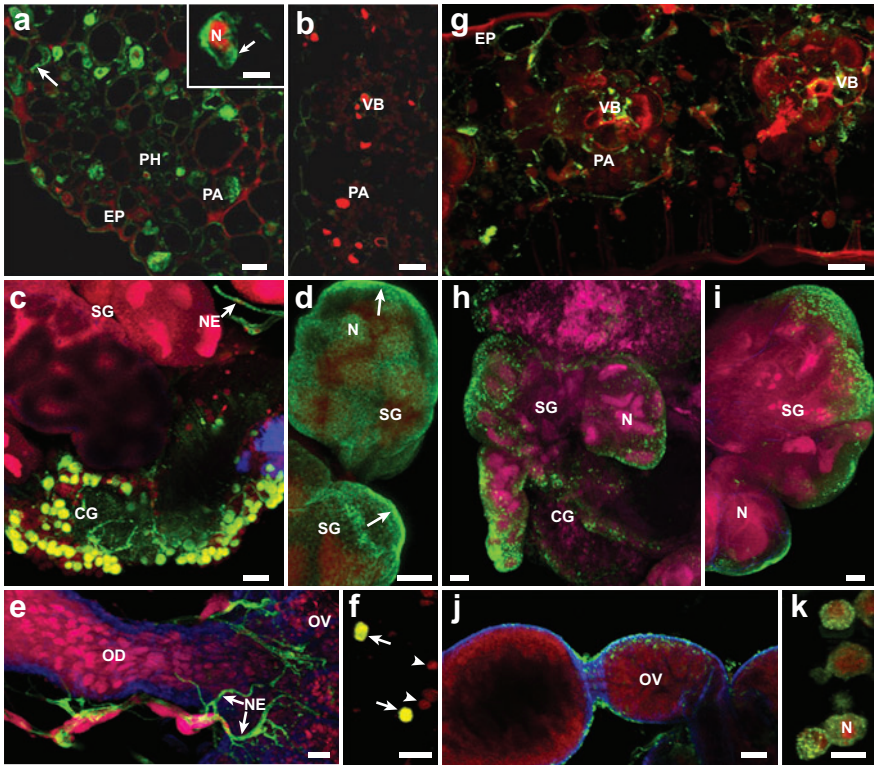

Figure 4

Immunofluorescence localization of MMV, Rhabdoviridae (*a-f*), and MStV, Tenuivirus (*g-k*) in sections of maize leaves and in whole-mount organs/tissues of their planthopper vector *P. maidis*. Polyclonal antibodies to either virus were used, followed by Alexa Fluor 488 (*green*) as secondary antibodies, and the nuclear stain propidium iodide (*red*). Thus, green/yellow fluorescence in this figure indicates presence of MMV/MStV particles. (*a*) & (*g*), Accumulations of MMV (*a*) and MStV (*g*) in sections of infected maize leaves; arrows in (*a*) indicate a few cytoplasmic (as opposed to the mainly perinuclear) accumulations of MMV. (*a*, inset) shows that MMV particles (green fluorescence) surround the nucleus (red fluorescence). (*b*) Control (uninfected) maize leaf section. (*c*) & (*h*), At 3-week post virus acquisition, the salivary glands (SG) are markedly infected with MStV (*h*) but not with MMV (*c*), whereas the compound ganglion (CG) is heavily infected with MMV but not with MStV. (*d*) & (*i*), At 4-week post virus acquisition, the salivary glands are infected with MMV (*d*) and with MStV (*i*); note that in the salivary glands MMV accumulates both around the nucleus (N) and near the cell periphery (*arrows*), whereas MStV appears mostly cytoplasmic. (*e*) Nerves (NE) of the ovarioles (OV) and oviduct (OD) infected with MMV; (*j*), the outer (follicular) cells of the ovarioles are infected with MStV; (*f*), Perinuclear accumulation of MMV in hemocytes (*arrows*); arrowheads indicate uninfected cells/nuclei. (*k*), Cytoplasmic accumulations of MStV in hemocytes. Other abbreviations: EP, epidermis, N, nucleus; NE, nerve; PA, parynchyma; PH, phloem; VB, vascular bundles. Scale bars, 20 μm except for the inset in (*a*) (5 μm).

to MMV transmission in *P. maidis*. In planthoppers, the esophagus and anterior diverticulum are sandwiched between the compound ganglionic mass and the salivary glands. Thus, Ammar & Hogenhout (10) postulated that MMV may bypass transmission barriers in *P. maidis* by moving from the midgut to the anterior diverticulum and esophagus, and from these organs to the salivary glands via the nervous system. This neurotropic route is somewhat similar to that of some vertebrate-infecting rhabdoviruses in dipteran hosts (45). Two other possible routes for arboviruses in their vectors were suggested by Romoser et al. (147) who reported that tracheae and visceral muscles may facilitate the movement of some arboviruses through the basal laminae of the mosquito midgut. These routes, as well as the possible role of the hemolymph, remain to be investigated for propagative plant viruses.

Tenuiviruses

The genus *Tenuivirus* includes four definitive members, including *Maize stripe virus* (MStV) and the type member *Rice stripe virus* (RSV), as well as eight tentative member viruses, all propagatively transmitted by planthoppers (Delphacidae, Hemiptera) (**Table 5**). Another tentative member, *Maize yellow stripe virus* (MYSV), is also transmitted propagatively by a leafhopper vector (Cicadellidae, Hemiptera) (15). Additionally, wheat yellow head virus (154) and two other viruses isolated from black spruce (33) are reported to have amino acid sequence similarities to Tenuiviruses, but none of these viruses has a known vector.

Tenuiviruses have plant host ranges normally limited to the family Poaceae, and may cause important diseases in several important crops particularly rice and maize in South East Asia and Latin America (reviewed in 50). Tenuiviruses are not seed transmitted and cannot normally be transmitted mechanically to plants except with VPI (105). Thus, transmission by insect vectors is essential for their spread and epidemiology in nature.

Tenuiviruses are apparently unique among plant viruses in having 4-6 segmented ribonucleoprotein particles (RNPs), each containing a single genomic RNA. Their genome size (ca 18-19 kb) is among the largest for plant viruses. The complete genomic sequences for several tenuiviruses have been determined (49, 139, 172). In purified preparations, the RNPs for various tenuiviruses may be filamentous, flexuous, spiral, branched, or circular; segments apparently have a length proportional to the size of the encapsidated RNA and a width of 3-10 nm. Virus-encoded nonstructural proteins have also been found in *Tenuivirus*-infected plants (50, 64). Falk & Tsai (50) suggested that *Tenuiviruses* and viruses in the genus *Phlebovirus* of the family *Bunyaviridae* have likely evolved from a common ancestor and retained a number of common molecular characteristics.

A tenuivirus-like segmented genome was detected for MYSV; the number and size of genome segments of MYSV were comparable to those of MStV. Complementary and conserved 5′ and 3′ termini similar to those of tenuiviruses and phleboviruses were detected for 4 of the MYSV segments. However, no nucleotide or amino acid sequence similarities with tenuiviruses were detected for the 5892 sequenced nucleotides of the estimated 18 kb genome of MYSV, and no cross hybridization was detected between MYSV and MStV (108). Based on these findings and the fact that MYSV is transmitted by a "cicadellid" leafhopper rather than "delphacid" planthoppers, Ammar & Peterschmitt (13) suggested that MYSV be placed in a new genus (*Cicatenuivirus*) in a newly erected family *Tenuiviridae* that contains the planthopper-borne tenuiviruses in the genus *Tenuivirus*. Because viruses within the *Rhabdoviridae*, *Reoviridae*, and *Geminiviridae* include plant viruses that are transmitted by different groups/families of hemipteran vectors, it would be interesting to look for vectors among these and other groups for tentative tenuiviruses for which no vectors have been reported so far, e.g., wheat yellow head virus (154) or the black spruce tenuivirus-like viruses (33).

Tenuiviruses can be acquired from infected plants by their vectors in acquisition periods ranging from 10 min to 4 h, whereas inoculation thresholds range from 30 s to nearly 1 h. These short acquisition and inoculation thresholds may indicate that tenuiviruses are not phloem-limited, which is also suggested by the fact that virus-specific inclusions were found in several leaf tissues, including the mesophyll, of host plants infected with MStV and RHBV (*Rice hoja blanca virus*; genus *Tenuivirus*) (8, 47). Recently, MStV-RNP-specific antigens were localized in various leaf tissues including the mesophyll by immunofluorescence microscopy (E-D. Ammar, unpublished data) (**Figure 4*g***). The proportion of transmitting insects and virus transmission efficiency can be increased by extending the acquisition access period of planthopper vectors on MStV-infected plants (7, 9). Latent periods of tenuiviruses in their vectors range from 1 to 36 days, but most are between 3 and 21 days. Virus inoculativity is retained by the vector up to 84 days post acquisition and probably throughout the vector's life, but efficiency of transmission usually declines with vector age (50, 78). Planthopper nymphs were reported as more efficient vectors than are adults for two strains of MStV, and females as more efficient vectors than males for RSV (50, 125, 174). Different populations of the same planthopper species may show variability in tenuivirus transmission efficiency, which can be increased or decreased by continuous selective breeding of active or inactive vectors (197). Transovarial transmission to a large proportion of the progeny of the vector (ca. 20%–100%) has been reported for most tenuiviruses (50, 78). RHBV also appears to be paternally transmitted (197). No transovarial or paternal transmission, however, has been reported for *Rice grassy stunt virus* or rice wilted stunt virus in their planthopper vector *N. lugens* (35), or for MYSV in its leafhopper vector *Cicadulina chinai* (15).

Tenuiviruses replicate in their host plants and insect vectors. Replication in the vector has been demonstrated by repeated transovarial passage of RHBV and RSV for 10 and 40 generations, respectively, in planthoppers (50), and by serological analysis of MStV and MYSV RNPs in their vectors. The percentage of *P. maidis* and *C. chinai* that were ELISA-positive for MStV or MYSV, respectively, increased over time corresponding with their ability to transmit these viruses to maize plants (15, 129). Also, the titer of MStV increased with time post acquisition in specific organs of *P. maidis*, including the salivary glands (129). In addition, MStV titer in *P. maidis* is positively correlated with transmission efficiency for three geographical isolates of MStV (7). The latent period was shorter in the more efficiently transmitted isolates, and the rate of transovarial transmission was positively correlated with oral acquisition/transmission efficiency in these isolates. This correlation was similarly positive in various populations of *Javesella pellucida* transmitting the European wheat striate mosaic virus (4). Together, these data suggest genetic control of tenuivirus transmission efficiency in these vectors.

Localization of tenuivirus RNP and noncapsid protein (NCP) in their plant and insect hosts has been challenging, probably because of the extremely thin and possibly variable shapes of RNPs. Using transmission electron microscopy (TEM) with immunogold labeling, most of the specific amorphous inclusions in leaf cells of MStV and RHBV-infected plants were labeled with antibodies to the NCP, but none was labeled with antibodies to RNP (8, 47, 48). In contrast, efforts to detect the NCP in MStV-infected planthoppers were unsuccessful (51). Similarly, the mRNA corresponding to the NCP-coding region of vRNA4 is abundant in MStV-infected plants, but was not detected in MStV-infected *P. maidis* (87). These results suggest that the replication strategy for MStV is different in the insect vs plant hosts. On the other hand, Chomchan et al. (38) detected accumulation of three nonstructural proteins (NSPs) encoded by *Rice grassy stunt virus* (RGSV) by Western blot analyses in both the plant and insect hosts: the 23-kDa p2 protein encoded on vRNA 2 (virus genomic strand); the 22-kDa p5 protein encoded on vRNA 5, the 21-kDa p6 protein encoded on vRNA 6. All

three proteins were detected in RGSV-infected rice leaf homogenates, and a large amount of p5 was detected in *Nilaparvata lugens* nymphs that were positive for the RNP protein, whereas small amounts of p2 and p6 were detected only in a subset of the N- and p5-positive insects. The authors suggested that p5 may have an essential role in virus infection in both plant and insect hosts, whereas p2 may function in plants as a cell-to-cell movement protein or silencing suppressor.

With RSV, four types of inclusion bodies were identified by immunofluorescence and immunogold microscopy of thin sections of infected leaves (101). As was the case with MStV and RHBV, most electron-dense, amorphous, semielectron-opaque inclusions associated with RSV contained only noncapsid protein (p4), but some contained the proteins p2, pc2-N, p3, and pc3 in addition to p4. In contrast, fibrillar, amorphous, semielectron-opaque inclusions contained only p4. Filamentous electron-opaque inclusions, which consist of pc2-N and p4, were found both in infected plant cells and in the midgut lumen and midgut epithelial cells of the planthopper vector *Laodelphax striatellus* (101). Suzuki et al. (165) had earlier reported immunogold labeling of amorphous/filamentous inclusions in the cytoplasm of midgut epithelial cells, salivary glands, and fat body of RSV-infected *L. striatellus*. In most of these in situ studies, no particular structure that resembles purified particles was labeled with the RNP antibodies. More recently, however, the spiral or circular filaments that are abundantly found in purified preparations and in the cell cytoplasm of MYSV-infected maize leaves (6) have been labeled with antibodies to MYSV-RNP (E-D. Ammar, A. Mahmoud & M. Peterschmitt, unpublished data). Like many plant-infecting viruses, RHBV encodes an RNA silencing suppressor, the NS3 protein. Recently, Hemmes et al. (77) reported that this protein is capable of suppressing RNA silencing in both plants and insect cells. Biochemical analyses showed that NS3 efficiently binds siRNA as well as miRNA molecules. Binding of NS3 is greatly influenced by the size of small RNA molecules, as 21 nucleotide (nt) siRNA molecules are bound more than 100 times more efficiently than 26 nt species. Competition assays suggested that the activity of NS3 binds siRNAs prior to strand separation during the assembly of the RNA-induced silencing complex. In addition, NS3 has a high affinity for miRNA/miRNA duplexes, indicating that its activity might also interfere with miRNA-regulated gene expression in both insects and plants (77).

The MStV N-protein has been detected by ELISA in MStV-infected plants and planthoppers and can accumulate in both (7, 51, 129); it was found in several organs of the planthopper *P. maidis* including the midgut, hindgut, Malpighian tubules, salivary glands, fat bodies, and reproductive organs (129). Similarly, the MStV mRNAs for each of the RNA2 ORFs have been detected in both MStV-infected plants and planthopper vectors (49). MStV was recently detected by immunofluorescence microscopy in various organs and tissues of its vector (**Figure 4**). It was detected in the salivary glands of *P. maidis* much earlier than the rhabdovirus MMV, which is also transmitted by *P. maidis* (see above), as early as 1 week post acquisition as compared to 3 weeks for MMV (10; S.A. Hogenhout, unpublished data). Additionally, infection of the nervous system of *P. maidis* by MStV appeared much less extensive (**Figure 4**) and occurred much later than that of the salivary glands. Thus, unlike MMV, MStV appears not to be neurotropic in *P. maidis*. MStV was localized in the cytoplasm in several tissues of the vector, including the esophagus, anterior diverticulum, midgut, hindgut, tracheae, muscles, and fat tissues. Additionally, it was found in the outer (follicular) cells of the ovarioles (**Figure 4*k***). Similarly, Suzuki et al. (165) reported that RSV, another *Tenuivirus*, was localized by immunogold TEM in the follicular cells of the ovarioles of its vector *L. striatellus*, and suggested that this may indicate transovarial transmission. However, MMV virions previously had been found by TEM in the follicular cells of ovaries of viruliferous *P. maidis*, which is not known to transmit MMV

transovarially (12). Thus, although some propagative viruses may be localized to some ovarian tissues, they are not necessarily transmitted transovarially.

Reoviruses

Members of family *Reoviridae* are segmented double-stranded RNA viruses with complex icosohedral complex virions composed of one, two, or three distinct layers (181). There are nine genera in the family. Viruses of genera *Orthoreovirus* and *Rotavirus* infect vertebrates only and are transmitted through feces and aerosols. Viruses of two other genera, *Orbireovirus* and *Coltivirus*, infect and replicate in both their vertebrate hosts and insect vectors. Members of the genus *Aquareovirus* infect aquatic vertebrates and crustaceans, and those of the genus *Cypovirus* infect lepidopteran, hymenopteran, and dipteran insects. Viruses in three genera, *Fijiviruses*, *Phytoreovirus*, and *Oryzavirus*, contain species that infect plants hosts, primarily members of the Poaceae (**Table 5**). Viruses in these genera also replicate in insect hosts and vectors, and are not seed transmitted. Insect transmission of the plant-infecting reoviruses is required in nature, and none can be transmitted mechanically, except by using VPI (105, 106).

The plant-infecting reoviruses have similarity to insect-infecting reoviruses such as leafhopper A virus and *P. maidis* virus. All plant-infecting reoviruses replicate in their insect vectors and are not considered to be seed transmitted. Effective acquisition and inoculation periods range from a few minutes to several days among the plant-infecting reoviruses, with shorter times being required for viruses that invade mesophyll as well as phloem tissues (127). Transovarial (vertical) transmission of reoviruses in insect vectors has been demonstrated for the fijiviruses *Fiji disease virus* (FDV), *Oat sterile dwarf virus* (OSDV), *Maize rough dwarf virus* (MRDV), and *Nilaparvata lugens virus* (NLV), and the phytoreoviruses *Rice dwarf virus* (RDV), *Wound tumor virus* (WTV) and *Rice gall dwarf virus* (RGDV). Phytoreoviruses are transmitted at higher rates (1.8%–100%) than fijiviruses (0.2%–17%). Although the rate of transmission is low for fijiviruses, FDV was transmitted transovarially for several generations and transmissivity was maintained for 6 years at 100% (84).

The brown planthopper *Nilaparvata lugens* is the host of NLV. Although the virus can be transmitted from insect to insect through rice, it does not replicate in the plant host (124) and is also transmitted vertically from insect to insect (132). This result stimulates two hypotheses about the evolution of the phytoreoviruses: either the virus, insect, and plant hosts coevolved or the viruses evolved as insect viruses that secondarily adapted to replicating in plant hosts. Several lines of evidence support the second hypothesis (126). The viruses replicate to higher titers in insect hosts than in plant hosts, some of the viruses are transmitted through insect eggs, but none is transmitted through seed. In addition, plants are inefficiently infected by single insects, and cytopathic effects of virus infection are greater in the plant than in the insect.

The phytoreoviruses, particularly RDV, are the best understood of the plant-infecting reoviruses. The three species in this genus infect both monocot and dicot plant hosts and are transmitted by cicadellid leafhopper vectors (**Table 5**). Phytoreoviruses have a double-shelled virion of ca. 70 nm. The atomic structure of the virion has been determined (91, 122), and indicates the positions of the inner P3 and outer P8 capsid proteins. The structure suggests that interactions between the major shell capsid proteins direct self-assembly of the virion (122), and in vivo self-assembly of the inner capsid protein (P3) and formation of double shells when coexpressed with P8 have been demonstrated (73, 74).

The RDV genome consists of 12 dsRNA segments that are 1066 to 4423 bp long. Proteins associated with all 12 segments of the RDV genome can be detected in both insect vector cells and rice, with much higher amounts of protein being present in the plant (163, 164). RGDV, WTV, and Tobacco leaf enation virus (TLEV) have a genomic organization similar to that of RDV, but individual genome segments

and encoded proteins have low sequence identity (117, 136, 198). Segments 1, 2, 3, 5, 7, 8, and 9 encode structural proteins. Segments 4, 6, 10, 11, and 12 encode nonstructural proteins. The roles of some of these proteins in the infection process are only partially defined at present.

P2, the major outer capsid protein that protrudes from the surface of the outer shell of the intact RDV virion, is involved in insect infection. The RDV outer shell is relatively unstable, and is lost during endocytosis. Intact RDV particles can enter and replicate in leafhopper (*Nephotettix cincticeps*) cells, but particles lacking a P2 protein due to chemical treatment or mutation cannot infect or attach to these cells and cannot be acquired by the leafhopper vector (134, 171, 194). The chemically treated virus that lacks P2 can replicate and is transmitted by the insect after injection into the hemocoel, but viruses carrying a mutant P2 do not replicate after hemocoel inoculation and are not transmitted. Thus, P2 may have a role in receptor recognition in the vector.

Interaction of the P8 outer-shell capsid protein with glycollate oxidase in rice or *Spodoptera* cells induces P8 localization to change from diffuse to punctate within 24 to 48 h after inoculation. The punctate P8 colocallizes with glycollate oxidase in peroxisomes (199). However, more work is needed to clarify the role of this localization in virus replication and viroplasm formation.

In plants, RDV P2 interacts with *ent*-kaurene oxidase-like proteins (199). These enzymes play a role in gibberellic acid synthesis in plants and their interaction with P2 is likely to be associated with symptom expression (e.g., gall formation). Inoculation of plants with RDV increases expression of defense and stress-related genes, and suppresses expression of genes required for cell elongation and photosynthesis (155).

Viroplasms formed shortly after RDV infection of host insect cells include the viral proteins Pns6, Pns11, and Pns12. Pns12 is a phosphorylated protein whose expression induces formation of viroplasm-like structures in nonhost insect cells. Immunocytochemical analyses identified virion core proteins (P1, P3, P5, and P7) at the interior of the inclusion bodies and the outer capsid proteins on the periphery (186). Viral inclusion bodies comprised of Pns10 form tubular structures ca. 85 nm in diameter and contain virus in insect host cells (185). In nonhost cells the tubules were associated with actin-based filopodea that protruded from the cell surface and penetrated neighboring cells. Recently, it was demonstrated that RDV enters insect cells through receptor-mediated endocytosis that is inhibited by drugs that disrupt clatherin activity (187). The Pns10 protein specifically binds actin, and formation of tubules as well as intercellular spread of RDV were inhibited by actin filament elongation-inhibiting drugs. The atomic structure of these tubules has been determined (92). The data suggest that the interaction of Pns10 with actin and the formation of the filopodia are important for virus spread in insects. Pns4 is associated with minitubular structures ca. 10 nm in diameter in viruliferous insects, similar to those formed in animal cells infected with bluetongue virus (185). The function of these minitubules in virus infection remains to be determined.

Similar to phytoreoviruses, fijiviruses have double-shelled icosahedral virions of 65–70 nm with a fragile outer shell. Fijiviruses incite hypertrophy and enations in their graminaceous hosts, and are transmitted by delphacid planthopppers. They are divided into five groups based on their insect and plant hosts (**Table 1**). *Rice black streaked dwarf virus* (RBSDV), MRDV, *Mal de Rio Cuarto virus* (MRCV), and FDV cause disease in rice and maize in Asia, Europe, South America, and Australia, respectively (142). Group 5 contains NLV, which infects the brown leafhopper but does not replicate in rice, the insect's breeding host (123, 124).

Complete genome sequences are available for FDV, MRCV, RBSDV, and NLV, and some information is available for MRDV and OSDV. For FDV, the 10 genomic segments encode 12 proteins. Of these, several structural proteins, including the B spike protein, have been identified. The RNA-dependent RNA polymerase

(RdRP) was identified based on homology with other viral polymerases (111). Functions have not yet been ascribed to proteins encoded by segments 4–8, and the importance of specific proteins in insect transmission has not been determined. Transmission of FDV from virus-tolerant plants is significantly lower than from susceptible cultivars (43). It was hypothesized that this lower rate of transmission slows virus spread in the field.

The least information is available on oryzaviruses. The two viruses in this genus have double-shelled virions 78–80 nm in diameter, and replicate in both their delphacid planthopper vectors and Poaceae hosts.

Marafiviruses

The genus *Marafivirus* includes three definitive members, including the type member *Maize rayado fino virus* (MRFV); all infect plant hosts in family Poaceae and all are persistently and propagatively transmitted by leafhoppers (Cicadellidae, Homoptera). Three other viruses that infect citrus and grapes appear to be related in genomic sequence to marafiviruses, but so far have no known vectors. MRFV has several experimental vector species in the genus *Dalbulus*, but *D. maidis* is the natural and most efficient one. MRFV causes epidemics in maize in Central America, where it often occurs in field infections in association with mollicutes (corn stunt spiroplasma and maize bushy stunt phytoplasma), which are transmitted by the same vector *D. maidis* (58, 128). Based on sequence analysis and the genetic distances among different geographic isolates of MRFV, the virus may have originated in Mexico and/or Guatemala and dispersed from there to the rest of the Americas (36).

Marafiviruses have icosohedral particles, 28–33 nm in diameter, and a single-stranded positive-sense RNA genome (58). The complete genome sequences of MRFV and *Oat blue dwarf virus* have been determined (46, 75). There are sequence similarities between these two viruses, and between the two viruses of the genus *Tymovirus*. Based on genomic structure and other similarities, Martelli et al. (110) suggested that the genus *Marafivirus* be included in family *Tymoviridae*, which does not include any leafhopper- or even hemipteran-borne viruses. The marafiviruses are not seed-borne and cannot be mechanically transmitted except by VPI (105, 107).

Marafivirus particles are most frequently observed in the phloem and vascular parenchyma of their poaceous hosts. Thresholds for *Marafivirus* acquisition and inoculation by leafhoppers range from several minutes to several hours, with longer acquisition periods resulting in higher transmission levels (20, 58). The latent period in the vector is 7 days or longer following acquisition from diseased plants, but injection of virus into the hemocoel reduced this period to 1 to 3 days and increased the transmission rate (20, 128). Nymphs are reported to be better vectors for MRFV than adult leafhoppers (128). Marafiviruses multiply in their vectors but are not transovarially transmitted (20, 65). Transmission rate of MRFV by *D. maidis* was increased several fold by selective breeding, but this enhanced ability dropped to normal rates after a few generations of random mating (58, 128).

SUMMARY POINTS

1. Of the ~700 plant viruses that are officially recognized by the ICTV more than 75% are transmitted by insect vectors, predominantly those of the Hemipteroid assemblage that includes aphids, whiteflies, leafhoppers, planthoppers, and thrips. In many cases, particularly in nonvegetatively propagated crops, insect transmission is obligatory for the plant virus, i.e., the insect vector is the only means of virus spread in nature.
2. The stylets of plant-feeding hemipteroid insects allow feeding from the plant phloem, xylem, and/or mesophyll cells, providing a route for uptake and inoculation of various plant viruses, including phloem-limited viruses.

3. The persistent viruses are subdivided into two subcategories, the persistent circulative (mostly nonpropagative) viruses and the persistent propagative viruses.
4. The movement and/or replication of persistent viruses in their insect vectors requires specific interactions between virus and vector components to overcome four major transmission barriers: (*i*) midgut infection barrier; (*ii*) dissemination (including midgut escape and salivary gland infection) barriers; (*iii*) salivary gland escape barrier; and (*iv*) transovarial transmission barriers.
5. The coat proteins of the nonenveloped persistent circulative viruses interact with insect-derived components, including GroEL homologues produced by aphid and whitefly bacterial endosymbionts, and various proteins that are involved in endocytosis and exocytosis pathways in insect vector gut and salivary gland cells.
6. Tissue tropism of the persistent propagative virus genera in their insect vectors varies greatly. Furthermore, these viruses are likely to infect the salivary glands either from the hemolymph or from other connecting tissues, such as tracheae, nerve cells, or muscle fibers.
7. The glycoproteins of enveloped viruses and the surface-exposed P2 protein of reoviruses are important for invasion of various insect vector tissues and hence for the successful infection of the salivary glands and subsequent introduction of virus into plants.

FUTURE PERSPECTIVES

Plant virus-related disease incidences, it is widely feared, will likely increase in the future. More intensive farming practices, in which crops are grown year round, may facilitate the buildup of both pathogen and vector reservoirs. Climate change may also allow for the spread and/or increases in some vector populations, resulting in more virus disease outbreaks in the more temperate regions of the world. Unlike aphids, leafhoppers and planthopppers are more abundant in regions without severe winters. These insects generally do not survive for long periods in areas with below-freezing temperatures, but several species can migrate for long distances. Indeed, the temperate regions in Europe and northern America that have experienced warmer winters in the past few years have simultaneously experienced more problems with insect-transmitted disease in various crops, ornamental flowers, and trees.

The decreased use of pesticides associated with organic farming practices and the deployment of transgenic crops that carry resistance against lepidopterans and beetles may indirectly lead to larger populations of virus-transmitting hemipteroids. Traditionally, investigators have focused on generating plant varieties with resistance to viruses. However, plant resistance to hemipteroid insects may also be useful for controlling diseases caused by persistently transmitted viruses (57, 150), and would have the added potential benefit of further reducing insecticide use for pest and disease control.

Still under investigation are questions particularly with regard to vector specificity and how plant viruses overcome various transmission barriers in their vectors. Still unresolved are the roles played in vector specificity by virus proteins, insect proteins, virus receptors, insect symbionts, as well as the role of the hemolymph and other tissues, e.g., the tracheae, visceral muscles, and nervous tissues, in the transmission process. Also, barriers to transovarial

or "vertical" transmission of viruses in their vectors remain grossly under-investigated. It is hoped, with the advent of new molecular technology, that such roles can be elucidated further and eventually exploited to combat plant viruses and their epidemics in economically important crops.

DISCLOSURE STATEMENT

The authors are not aware of any biases that might be perceived as affecting the objectivity of this review.

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Errata

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